Autonomic nervous involvement in stress-induced ACTH secretion

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SUMMARY

The purpose of this study has been to investigate the influence of the autonomic nervous system on the secretion of the adrenocorticotrophic hormone (ACTH) from the pituitary gland following acute stress in rats.

First a survey is given of the endocrinological research on the stress mechanism (I, 2), in which special attention is paid to the stress concept of SELYE (I, 2, A), and the current views concerning the regulation of the ACTH secretion by the systemic blood level of the adrenocortical hormones (I, 2, B), by the adrenomedullary hormones (I, 2, C) and by humoral factors elaborated by the hypothalamus (I, 2, D). It is concluded that the experimental work in the endocrinological field indicates that the central nervous system, particularly the diencephalon, exerts a controlling function on the secretory activity of the hypophysis, but that the exact mechanism of this regulation is still poorly understood.

Then a review is presented of REILLY's concept of the "irritation syndrome", which is caused by noxious stimuli irritating the peripheral sympathetic nervous system (I, 3, A). The French work indicates that the sympathetic irritation is centrally induced, and suggests that the reticular structures in the brain stem are indispensable for these reactions (I, 3, B).

After a comparative discussion of the concepts of SELYE and REILLY (I, 4), more attention has been paid to the role of the central nervous system in stress reactions (I, 5). The main investigations on the diencephalic regulation of the vegetative, defensive and affective functions of the body (HESS), and on the effect of noxious stimuli and emotional disturbances on the hypothalamic balance (GELLHORN) have been reviewed. This work shows that the diencephalon represents the integrative and controlling structure for the homeostatic reactions to disturbance of the somatic and psychic equilibrium of the organism, which reactions are mainly mediated through the autonomic nervous system. The sympathetic and parasympathetic divisions of this system are represented in respectively the posterior and the anterior part of the hypothalamus.

Thus the influence of the autonomic nervous system on the ACTH secretion may occur at two levels: the peripheral level, through the peripheral autonomic innervation of the pituitary gland; and the central level, through a direct action of the diencephalic autonomic structures on the release of stimuli (nervous and/or humoral) descending via the hypophysial stalk (I, 6). Consequently, the experimental work is concerned with aspects of the peripheral and the central effects, i.e. with the influence of

faradic irritation of the sympathetic hypophysial innervation on the ACTH release (II), and with the influence of hypothalamic lesions on the ACTH secretion following stress (III).

Some anatomical data on the innervation of the pituitary gland are provided in II, 1. Next the histological changes in the pars distalis of the hypophysis due to sympathetic irritation are described (II, 2, A). The ACTH releasing capacity of faradic irritation is measured in animals, in which a central activation is blocked by pretreatment with corticosteroids (II, 2, B). It is concluded that the tissular damage which results from sympathetic irritation and causes a direct contact between the glandular parenchyma and the blood stream, cannot explain the mechanism of the ACTH release (II, 3).

The experimental work on the regulation of the ACTH secretion with the aid of lesions placed in the hypothalamus has been discussed (III, 1). The present experiments in rats were concerned with a comparison of the effects of three different hypothalamic lesions on the ACTH secretion following the application of acute stress. These lesions destroyed the tuberal area (including the median eminence), the anterior ("trophotropic" or parasympathetic) part, or the posterior ("ergotropic" or sympathetic) part of the hypothalamus. The effect of traumatic, painful, systemic and emotional stimuli on the ACTH secretion was measured indirectly on the adrenal ascorbic acid depletion in these three groups of lesioned animals and in normal intact animals (III, 3).

It can be inferred from the results that the tuberal lesion abolished the effect of all types of stress, except for histamine. The anterior lesion diminished the effect of somatic (= traumatic and systemic) stimuli, and abolished the effect of psychic (= emotional and painful) stimuli. Psychic stimuli were also abolished by posterior lesions, but somatic stimuli were potentiated by these lesions. The effect of ACTH and Pitressin remained unaffected in all lesioned groups (III, 4).

A hypothesis is advanced concerning the mechanism of ACTH release in somatic and psychic stress. Somatic stimuli are supposed to activate the reticular formation of the brain stem, from where impulses pass on to the tuberal area of the hypothalamus, causing the release of the transmitter substance which activates the adenohypophysis for ACTH secretion. Psychic stimuli are thought to be conducted from the cerebral cortex via the limbic system and the descending column of the fornix to the posterior hypothalamus. Activation of this area may result in an activation of the reticular formation, and also in a stimulation of the adrenal medullary secretion, resulting in the release of adrenaline, which hormone on its turn causes an ACTH discharge (IV, see also Fig. 8).